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PAPER

Early anthropometric measures and reproductive factors as predictors of body mass index and obesity among older women

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OBJECTIVE: To examine whether early anthropometric measures and reproductive factors were associated with body mass index (BMI), overweight, and obesity.
DESIGN: Cross-sectional, observational study.
SUBJECTS: In all, 18 109 healthy women who participated in the Swedish Mammography Cohort aged 49–83 y.
MEASUREMENTS: Early anthropometric (birthweight and body shape at age 10 y) and reproductive (age at menarche, age at the birth of the first child, and parity) variables were our predictors and current BMI, overweight (BMI 25–29.99 kg/m^2), and obesity (BMI $\geq$30 kg/m^2) were our outcomes.
RESULTS: In multivariate-adjusted polytomous logistic regression analysis, risk of overweight and obesity increased with increasing body shape at age 10 y and decreased with increasing age at menarche and age at first birth (P for trend <0.0001). A U-shaped relation with birthweight was observed. In our tests for effect modification of the relation with overweight/obesity (ow/ob; BMI $\geq$25 kg/m^2), we detected significant interactions between body shape at 10 y and age ($P<0.0001$); body shape at 10 y and physical activity ($P<0.0001$); age at first birth and smoking ($P=0.02$); and parity and physical activity ($P=0.004$). The increased risk of ow/ob among women who reported a larger childhood body shape was reduced as women moved from the lowest to highest quartile of physical activity in adulthood. Likewise, the increasing risk of ow/ob among women with greater parity was reduced with increased physical activity.
CONCLUSION: Early anthropometric measures and reproductive factors are significantly associated with BMI, overweight, and obesity among older women. The effects of childhood body weight, age at first birth, and parity may be modified by adult lifestyle choices, as well as age.
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Keywords: BMI; childhood; reproductive; birthweight; physical activity

Introduction

Overweight and obesity, as measured by body mass index (BMI) 25–29.99 and $\geq$30 kg/m^2, respectively, are increasingly common in Europe, especially among women.[1] Women have a greater percentage of body fat than men to prepare for child-bearing and body composition shifts throughout the reproductive cycle. In addition, fluctuations in hormone concentrations throughout the life cycle predispose women to weight gain and hormonal changes during the menstrual cycle affect energy intake, dietary patterns, and alter 24-h energy expenditure.[2] Women also have lower energy expenditure for a given fat mass compared to men.[3]

The initiation and tracking of obesity may begin early in life. A systematic review of longitudinal observational studies confirmed a positive association between birthweight and BMI in both children and adults.[4] However, studies on the relationship to BMI and obesity among older adults are sparse. A recent review of the relation of birthweight with adiposity and fat distribution in later life found inconsistent results among older adults, although many of the studies included had a small sample size and inadequate adjustment for potential confounders.[5] A 40-y follow-up of overweight children aged 0–16 y at baseline found that being overweight

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in adolescence was the most significant predictor of adult weight,[6] and a review indicates approximately 50% of adolescents with a BMI at the 95th percentile became obese adults.[7]

As women age, reproductive factors may also impact their risk of obesity, including the age at the onset of sexual maturity with menarche in adolescence and choices concerning child-bearing in young adulthood (ie when to begin having children and how many children to have). Earlier menarche was associated with higher BMI in young adults[8] and the relation was stronger among premenopausal women than postmenopausal women in one study.[9] Earlier age at first birth was associated with overweight among postmenopausal women[10,11] and parity may be related to overweight among women either through high gestational weight gain within an individual pregnancy or cumulative weight gain following successive pregnancies.[12] Parity was associated with an increase in body weight of 0.55 kg per live birth (0.09 kg/y) among women 18–50 y, but the effect was weak when adjusted for age.[13] These studies indicate that both age and menopausal status need to be considered when examining weight gain among women.

Although many studies have examined independently the effects of some of these early anthropometric and reproductive factors on BMI and obesity, few of them examined simultaneously their effects. Our objective was to determine whether early anthropometric measures birthweight and body shape at age 10 y and reproductive factors age at menarche, age at first birth, and parity were independent predictors of adult BMI among a sample of Swedish women. We also examined whether the effects of these variables were modified by age or lifestyle choices such as physical activity and smoking.

We hope that our study will help elucidate whether risk factors that occur earlier in life in childhood and young adulthood remain significant risk factors for obesity later in life as women age. Examining whether effects of early exposures are modified by age, physical activity, and smoking may further our understanding of the etiology of obesity. Importantly, our study may also suggest targets for prevention, which could help to reduce the burden of obesity.

Methods

Study design and participants

Data analyzed in this study were from the Swedish Mammography Cohort (SMC), a population-based mammography screening program introduced in 1987 to Västmanland and Uppsala counties in central Sweden. All women born between 1914 and 1948 living in these two counties were invited to the screening ($n = 90\,903$) and received a six-page questionnaire that included items about anthropometric, reproductive, sociodemographic, and dietary factors. Among them, 66 651 (74%) completed the questionnaire and agreed to participate; these methods are further described elsewhere.[14] In brief, exclusions from the study included the following: questionnaires with missing ($n = 707$) or incorrect ID numbers ($n = 413$); questionnaires lacking a date ($n = 608$); women who moved out of the study area ($n = 79$); and women who died during the study period, but were missing a date of death ($n = 16$). A second questionnaire was mailed to 58 055 participants in the SMC participants in October 1997, of whom 39 008 (67%) responded.

As the first questionnaire did not include measures on the main exposure variables tested in this analysis, only data from the second questionnaire in 1997 were used in this study. Our data set only included women with complete data on both outcome and exposures. We first excluded women who did not provide information on height or weight ($n = 6532$) or whose values for weight, height, or BMI appeared implausible ($n = 310$). We also excluded subjects with missing data on any of our main exposure variables (birthweight, body shape at age 10 y, age at menarche, age at first birth, and parity) ($n = 14\,057$). After all exclusions, 18 109 subjects were included in the analysis.

Measurements

Early anthropometric measures (birthweight and body shape at age 10 y) and reproductive factors (age at menarche, age at the birth of the first child, and parity) were the main exposure variables in this study, all of which were self-reported on the questionnaire. Women self-reported their birthweight in five mutually exclusive categories ranging from <1500 to >5000 g. Subjects recalled their body shape ('How was your figure at 10 y of age?') by choosing among seven figures of increasing body size, ranging from smallest (1) to largest (7). Recall of body size at age 10 y using somatotype pictograms with body size at the same age showed high validity ($r = 0.70$) among older white females in the United States (mean age 73.1 y)[15] and a population-based validation study among Swedish woman aged 61–66 y found correlations between BMI from school records and adult report of somatotype at ages 7 and 13 y were 0.6 and 0.7, respectively (A Wolk, personal communication). Age at menarche was recalled by answering the question 'How old were you when your period started?' and parity was determined by asking study participants to report 'How many children have you had?' Women were also asked to recall their 'age at the birth of the first child'.

Data on additional covariates were also collected from the questionnaire, including menopausal status (yes/no), use of hormone replacement therapy at menopause (yes/no), smoking habit (current, previous, or never), childhood home (city/suburb, medium-sized city, smaller town/community, or country), and education/all schools attended (compulsory school, vocations/girls' school, junior secondary school, secondary school, university/college, and other training). Education was collapsed into four mutually exclusive categories of primary school, high school, university, and

other training. Daily physical activity was computed in metabolic equivalents (METs; kcal/kg × h) using a short questionnaire that inquired about physical activity and inactivity at work and at home as well as leisure-time activity and hours spent sleeping. The questionnaire showed reasonable validity ($r = 0.56$) when validated using structured 7-day physical activity diaries, as further described elsewhere.[16]

Statistical analyses

BMI (kg/m^2) was calculated for each subject from self-reported weight and height. High validity has been observed for self-reported height ($r = 1.0$), weight ($r = 0.9$), and BMI ($r = 0.9$) compared to actual measurements among Swedish women.[17] Sample characteristics were described using means and standard deviations (s.d.) for continuous variables and frequencies (number and percent) for categorical variables, both for the whole cohort and also stratified into three BMI groups of normal BMI (BMI <25 kg/m^2), overweight (BMI 25–29.99 kg/m^2), or obese (BMI ≥ 30 kg/m^2), following international cutpoints.[18]

Regression analyses were performed to examine the relation between early anthropometric and reproductive variables and BMI, overweight, and obesity. In the main analyses, our exposure variables were transformed to categorical variables using indicator variables and the reference group was the group with the highest percentage of subjects. In addition, because of the small numbers of subjects with birthweight <1500 or ≥ 5000 g, birthweight was collapsed into three categories of low birthweight (<2500 g,) normal birthweight (2500–3999 g), and high birthweight (≥ 4000 g).

In our first analysis, multiple linear regression was used to predict BMI and a trend test was performed. The second analysis used polytomous logistic regression, in which the dependent variable (BMI group) was treated as a categorical variable with three levels (normal weight, BMI <25 kg/m^2; overweight, BMI 25–29.99 kg/m^2; obesity, BMI ≥ 30 kg/m^2). In our first model, we treated BMI group as an ordinal variable, which assumes proportional odds. However, the score test for the proportional odds assumption was significant ($P<0.0001$), indicating that this assumption could not be met, and a partial proportional odds model showed similar results ($P<0.0001$). Therefore, we selected a generalized logits model, which estimates separate regression coefficients for each level of the variable (ie overweight relative to normal weight and obese relative to normal weight). In all regression analyses, all of our predictor variables were included in one model, which also included additional sociodemographic (age, smoking, childhood home, and education) and later reproductive (menopausal status and use of hormone replacement therapy) risk factors for overweight.

Our secondary analyses checked for interaction effects. We tested whether the effects of our main exposure variables (birthweight, body shape at age 10 y, menarche, age at first birth, and parity) were modified by age, physical activity, or smoking status; we created crossproduct terms for each of these relations (eg age × birthweight, physical activity × body shape at age 10 y, etc) and added them to the final, multivariate-adjusted model to see if they were significant.

Results

Sample characteristics are presented for the whole cohort and are stratified into BMI groups in Table 1. The mean age ($\pm$s.d.) among study participants was 59.4 ± 8.5 y with a mean BMI ($\pm$s.d.) of 25.0 ± 3.9 kg/m^2. The majority of subjects were postmenopausal (84%), never smokers (57%), grew up in a rural area (46%), and had a primary school education (43%). Compared to women who were normal weight or overweight, a higher percentage of obese women were never smokers and a lower percentage of obese women were university educated.

Table 2 presents results from the linear regression analysis. A larger BMI was observed both for subjects with a birthweight <2500 and ≥ 4000 g compared to those with birthweight 2500–3999 g. BMI was positively associated with body shape at age 10 y and inversely associated with age at menarche and age at first birth. Compared to a parity of 2, only the top category of parity (≥ 5) was significantly ($P<0.0001$) related to BMI. A significant ($P<0.05$) linear trend was observed for all variables but birthweight.

Polytomous logistic regression results are presented in Table 3. Relations with overweight and obesity were in the same direction as in the linear regression analysis. The magnitude of risk was generally greater for obesity than for overweight, although in many cases confidence intervals overlapped. Women of birthweight ≥ 4000 g had a 45% increase in odds of obesity (CI: 1.25, 1.68), while the relation for overweight was not significant (OR = 1.04; CI: 0.94, 1.16). The odds of obesity increased with increasing body shape at age 10 y, and women who reported their body shape at age 10 y ≥ 5 were 2.16 (CI: 1.90, 2.47) more likely to be overweight and 5.40 (CI: 4.52, 6.45) more likely to be obese compared to those who ranked themselves as a 2. Parity was not significantly related to odds of overweight, while a parity of 4 or more significantly increased the odds of obesity ($P<0.05$).

In our tests for effect modification, we detected significant interactions between body shape at 10 y and age ($P<0.0001$); body shape at 10 y and physical activity ($P<0.0001$); age at first birth and smoking ($P=0.02$); and parity and physical activity ($P=0.004$). We therefore performed additional stratified analyses for each of these relations (Figure 1). For ease in presentation and to maximize statistical power, we collapsed the overweight and obese subjects into one group, overweight/obese (ow/ob; BMI ≥ 25 kg/m^2), and used a binary logistic regression model in each set of stratified analyses to predict odds of ow/ob. We also changed the

Table 1 Sample characteristics of 18 109 women participating in the Swedish Mammography Cohort, entire sample and stratified by BMI

Sample characteristic[a]	*Entire sample (n = 18 109)*	*BMI < 25 kg/m²* *(n = 10 169)*	*BMI 25–29.99 kg/m²* *(n = 6057)*	*BMI ≥ 30 kg/m²* *(n = 1883)*
Age (y), mean ± s.d.	59.4 ± 8.5	58.7 ± 8.5	60.2 ± 8.5	60.3 ± 8.1
BMI (kg/m²), mean ± s.d.	25.0 ± 3.9	22.4 ± 1.7	27.0 ± 1.4	33.0 ± 3.0
Birthweight (g), N (%)				
<1500	81 <1	34 <1	36 <1	11 <1
1500–2499	1560 (9)	832 (8)	535 (9)	193 (10)
2500–3999	14,356 (79)	8191 (81)	4791 (79)	1384 (74)
4000–4999	1923 (11)	1031 (10)	629 (10)	263 (14)
≥5000	179 (1)	81 (1)	66 (1)	32 (2)
Body shape at 10 y, N (%)[b]				
1	4491 (25)	2832 (28)	1332 (22)	327 (17)
2	5178 (29)	3246 (32)	1575 (26)	357 (19)
3	3974 (22)	2173 (21)	1400 (23)	401 (21)
4	2928 (16)	1330 (13)	1150 (19)	448 (24)
≥5	1538 (8)	588 (6)	600 (10)	350 (19)
Age at menarche (y), N (%)				
≤11	1916 (11)	909 (9)	680 (11)	327 (17)
12	3566 (19)	1841 (18)	1296 (21)	429 (23)
13	4853 (27)	2727 (27)	1638 (27)	488 (26)
14	4344 (24)	2545 (25)	1431 (24)	368 (20)
≥15	3430 (19)	2147 (21)	1012 (17)	271 (14)
Age at first birth (y), N (%)				
17–19	2879 (16)	1400 (14)	1066 (18)	413 (22)
20–22	4811 (27)	2535 (25)	1698 (28)	578 (31)
23–25	4589 (25)	2676 (26)	1500 (25)	413 (22)
26–28	3270 (18)	2015 (20)	987 (16)	268 (14)
≥29	2560 (14)	1543 (15)	806 (13)	211 (11)
Parity, N (%)				
≤1	2806 (16)	1595 (16)	944 (15)	267 (14)
2	8484 (47)	4908 (48)	2773 (46)	803 (43)
3	4597 (25)	2534 (25)	1557 (26)	506 (27)
4	1481(8)	783 (8)	509 (8)	189 (10)
≥5	741 (4)	349 (3)	274 (5)	118 (6)
Smoking, N (%)				
Never	9025 (57)	4869 (55)	3142 (59)	1014 (61)
Previous	4378 (27)	2383 (27)	1519 (28)	476 (28)
Current	2550 (16)	1649 (18)	715 (13)	186 (11)
Childhood home, N (%)				
Rural	8215 (46)	4340 (43)	294 (49)	931 (50)
Suburb	2204 (12)	1314 (13)	679 (12)	211 (11)
City	2868 (16)	1694 (17)	914 (15)	260 (14)
Town	4630 (26)	2717 (27)	1453 (24)	460 (25)
Education, N (%)				
Primary school	7777 (43)	3938 (39)	2866 (47)	973 (52)
Secondary school	1587 (9)	999 (10)	463 (8)	125 (7)
University	4236 (23)	2814 (27)	1133 (19)	289 (15)
Other training	4471 (25)	2396 (24)	1586 (26)	489 (26)
Physical activity (METs/day), mean ± s.d.	42.4 ± 4.7	42.5 ± 4.6	42.5 ± 4.8	41.4 ± 5.0
Postmenopausal, *N* (%)	14 234 (84)	7871 (83)	4849 (86)	1514 (87)
Hormone replacement therapy (ever users), *N* (%)	6840 (43)	4020 (45)	2187 (41)	633 (38)

[a]Sample sizes for covariates are <18 109 due to missing data, as follows: Menopausal status (*n* = 16 899), hormone replacement therapy (*n* = 16 095), smoking (*n* = 15 953), childhood home (*n* = 17 917), education (*n* = 18 071), and physical activity (*n* = 14 853). [b]Body shape at age 10 y responses range from 1 to 7, where 1 represents the smallest body size and 7 represents the largest.

Table 2 Multivariate linear regression analysis predicting BMI among 18 109 women participating in the Swedish Mammography Cohort[a]

Exposure variable	*BMI β (s.e.)*	P *for trend*
Birthweight (g)		0.11
<2500	0.27 (0.10)*	
2500–3999	0—Reference	
≥4000	0.41 (0.09)**	
Body shape at age 10 y[b]		<0.0001
1	0.15 (0.08)	
2	0—Reference	
3	0.59 (0.08)**	
4	1.47 (0.08)**	
≥5	2.32 (0.11)**	
Age at menarche (y)		<0.0001
≤11	0.95 (0.08)**	
12	0.39 (0.08)**	
13	0—Reference	
14	−0.27 (0.08)*	
≥15	−0.35 (0.08)**	
Age at first birth (y)		<0.0001
17–19	0.35 (0.09)**	
20–22	0—Reference	
23–25	−0.44 (0.08)**	
26–28	−0.63 (0.09)**	
≥29	−0.65 (0.09)**	
Parity		<0.0008
≤1	0.04 (0.08)	
2	0—Reference	
3	0.09 (0.07)	
4	0.19 (0.11)	
≥5	0.61 (0.14)**	

β = beta coefficient; s.e. = standard error. [a]All predictor variables are tested in the same model and models are further adjusted for age, smoking, childhood home, education, hormone use, and menopausal status. [b]Body shape at age 10 y responses range from 1 to 7, where 1 represents the smallest body size and 7 represents the largest. *$P<0.05$. **$P<0.0001$.

Table 3 Polytomous logistic regression analysis predicting overweight (BMI 25–29.99 kg/m^2) or obesity (BMI ≥30 kg/m^2) among 18 109 women participating in the Swedish Mammography Cohort[a]

Exposure variable	*Overweight OR (CI)*[b]	P *for trend*	*Obesity OR (CI)*[b]	P *for trend*
Birthweight (g)		0.29		0.38
<2500	1.13 (1.01, 1.27)		1.43 (1.21, 1.70)	
2500–3999	1.00—Reference		1.00—Reference	
≥4000	1.04 (0.94, 1.16)		1.45 (1.25, 1.68)	
Body shape at 10 y[c]		<0.0001		<0.0001
1	0.95 (0.87, 1.04)		1.02 (0.87, 1.20)	
2	1.00—Reference		1.00—Reference	
3	1.31 (1.20, 1.44)		1.64 (1.40, 1.91)	
4	1.78 (1.61, 1.97)		2.98 (2.54, 3.48)	
≥5	2.16 (1.90, 2.47)		5.40 (4.52, 6.45)	
Age at menarche (y)		<0.0001		<0.0001
≤11	1.25 (1.11, 1.41)		1.89 (1.60, 2.23)	
12	1.19 (1.08, 1.31)		1.31 (1.13, 1.51)	
13	1.00—Reference		1.00—Reference	
14	0.93 (0.85, 1.02)		0.81 (0.70, 0.95)	
≥15	0.80 (0.72, 0.88)		0.75 (0.63, 0.88)	
Age at first birth (y)		<0.0001		<0.0001
17–19	1.16 (1.05, 1.29)		1.30 (1.12, 1.51)	
20–22	1.00—Reference		1.00—Reference	
23–25	0.88 (0.80, 0.96)		0.74 (0.64, 0.85)	
26–28	0.77 (0.70, 0.86)		0.65 (0.55, 0.77)	
≥29	0.83 (0.74, 0.93)		0.67 (0.55, 0.80)	
Parity		0.60		<0.0012
≤1	1.05 (0.95, 1.16)		1.07 (0.91, 1.25)	
2	1.00—Reference		1.00—Reference	
3	1.04 (0.96, 1.13)		1.12 (0.99, 1.28)	
4	1.02 (0.90, 1.15)		1.22 (1.02, 1.47)	
≥5	1.15 (0.97, 1.37)		1.60 (1.26, 2.02)	

[a]All predictor variables are tested in the same model and models are further adjusted for age, smoking, childhood home, education, hormone use, and menopausal status. Normal weight (BMI <25 kg/m^2) is the reference group. [b]OR = odds ratio; CI = 95% confidence interval. Confidence intervals that do not include 1 are statistically significant ($P<0.05$). [c]Body shape at age 10 y responses range from 1 to 7, where 1 represents the smallest body size and 7 represents the largest.

reference group in each analysis to denote the lowest or highest risk group to present the clearest picture of the associations.

Compared to women in the lowest age group with the smallest body shape at age 10 y, odds of ow/ob increased for increasing body shape at age 10 y, with higher risk seen in older age groups (panel a). Panel b shows that the risk of ow/ob increases as body shape at age 10 y increases across all quartiles of physical activity, but the effect of larger early childhood body shapes (4 and ≥5) on ow/ob is smaller in the higher quartiles of physical activity. Compared to never smokers whose age at first birth was 20–22 y, previous smokers had a similar, increased risk of ow/ob, while current smokers were at a significantly reduced risk of ow/ob across all categories of age at first birth (panel c). Women in the lowest quartile of physical activity were at an increased risk of ow/ob across all levels of parity, with the highest risk among women with parity ≥5; women with parity ≥5 were not at increased risk of ow/ob in quartiles 2, 3, or 4 (panel d).

Discussion

In this study, we found that early anthropometric measures and reproductive factors were associated with BMI, overweight, and obesity among older women representative of the Swedish population. Our study makes several important contributions to the literature. First, our study is a large, population-based sample that provided adequate power to detect small effects and our study design elucidated interaction effects with age, physical activity, and smoking. Second, we showed that early anthropometric and reproductive factors were independent of each other and remained significant when tested together in the same model. We were also able to adjust for additional risk factors for obesity

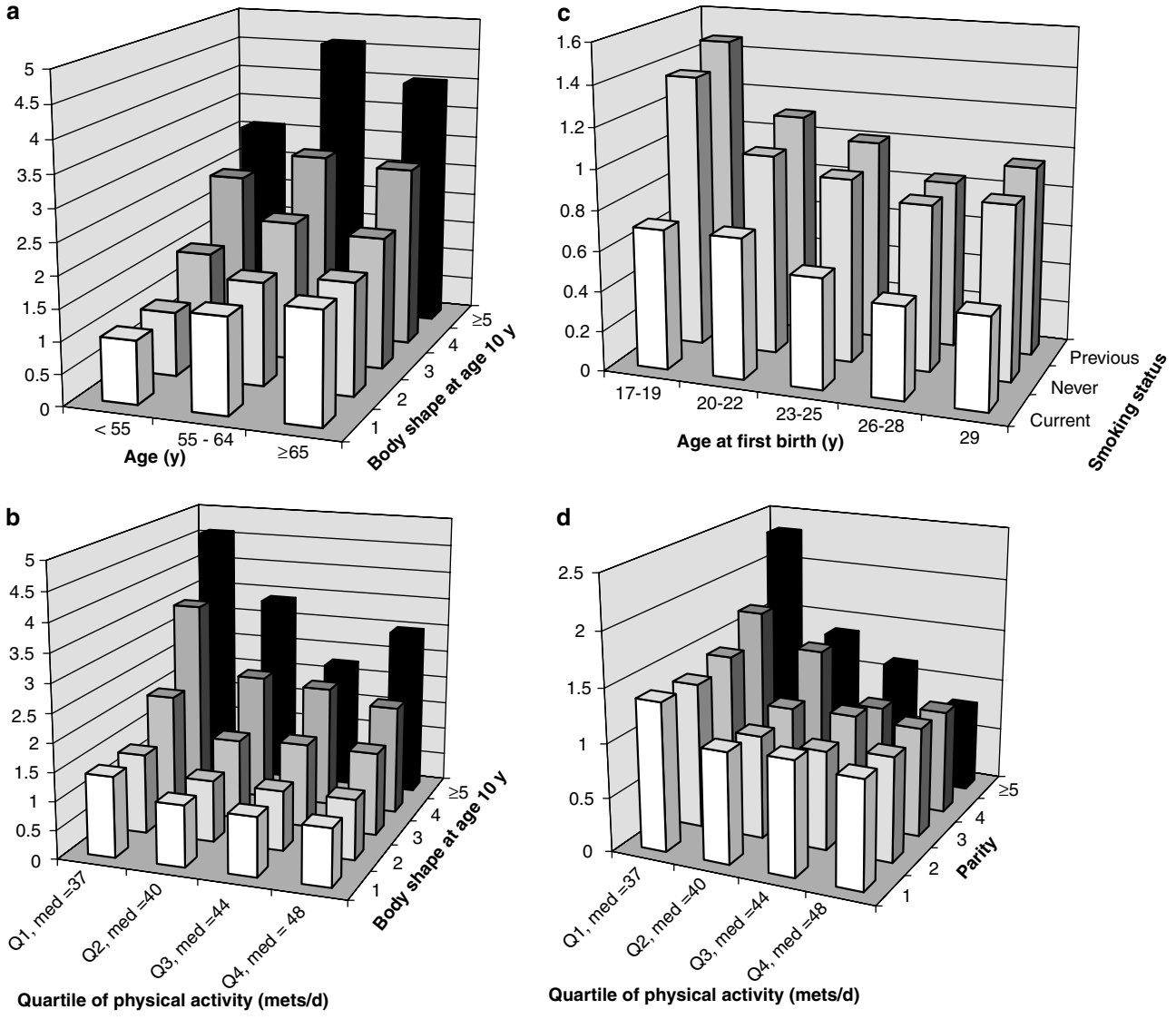

Figure 1 Interaction effects on the risk of overweight/obesity (BMI $\geq 25\,kg/m^2$) for body shape at age 10 y by age (a); body shape at age 10 y by physical activity (b); age at first birth by smoking (c); and parity by physical activity (d). Normal weight (BMI $< 25\,kg/m^2$) is the reference group for all analyses. All predictor variables are tested in the same model and models are multivariate adjusted. (a) Odds ratios (OR) showing the association between body shape at age 10 y and overweight/obese (BMI $\geq 25\,kg/m^2$), stratified by age group (reference group = age < 55 y, body shape = 1). ORs for each strata are significantly different ($P < 0.05$) than the reference, except for those age < 55 y, body shape = 2. (b) ORs showing the association between body shape at age 10 y and overweight/obese (BMI $\geq 25\,kg/m^2$), stratified by quartile of physical activity (reference group = quartile 4, body shape = 1). ORs for each strata are significantly different ($P < 0.05$) than the reference, except for those in Q2, body shapes 1 and 2; Q3, body shapes 1 and 2; and Q4, body shape 2. (c) ORs showing the association between age at first birth and overweight/obese (BMI $\geq 25\,kg/m^2$), stratified by smoking status (reference group = never smokers, age 20–22 y). ORs for each strata are significantly different ($P < 0.05$) from the reference, except for previous smokers 20–22 y, never smokers 23–25 y, and previous smokers 23–25 y. (d) ORs showing the association between parity and overweight/obese (BMI $\geq 25\,kg/m^2$), stratified by quartile of physical activity (reference group = Q4, parity ≤ 1). ORs are significantly different ($P < 0.05$) from the reference only for Q1, all parity groups, and Q2, parity = 4.

in our models, including smoking habit,[19] hormone use,[20] menopausal status,[21] childhood home,[22,23] and education.[24] Furthermore, although our study was cross-sectional in that the exposures and outcomes were assessed at the same point in time, our design approximates a prospective design in that all of the exposures considered here occurred before the outcomes. Of additional note is that our exposure variables were more strongly related to obesity than to overweight in a polytomous regression model, which may suggest a different trajectory of weight gain for women who are obese compared to those who are overweight. This finding requires more research.

In our study, both low and high birthweight were significantly associated with BMI, overweight, and obesity, indicating a U-shaped relation. A recent review confirms that both linear and U- or J-shaped relations between birthweight and BMI have been observed, although the evidence for a significant relation is less consistent for middle-aged subjects.[5] Our study is among only a few[25–27] that observed a significant relation between birthweight and BMI or obesity among older adults (>50 y), while other studies of similar age[28,29] saw no relation. Inconsistent results may be explained by inadequate adjustment for potential confounders, although an earlier review[4] states that the relation between birthweight and BMI in children and adults remained inconsistent in studies that adjusted for gestational age, parental fatness, or social group. Our findings for birthweight may be modified if we had been able to include these potential confounders in our analysis. An alternative explanation for inconsistent findings between birthweight and adiposity may be that birthweight is more significantly associated with lean mass rather than fat mass or BMI among older adults.[30,31] Of additional interest is that the association between birthweight and BMI may be different for men than for women,[26,32,33] which warrants further investigation.

Body shape at age 10 y was a significant predictor of BMI, overweight, and obesity, which is consistent with findings from two longitudinal studies.[34,35] In another longitudinal study, however, only 18% of obese women and 17% of obese men at age 33 y had been obese at age 7 y; thus, the authors concluded that prevention strategies should be population based because most obese adults could not be identified from their childhood BMI.[36] In addition, a 50 y follow-up of BMI from childhood to middle age showed no correlation between childhood and adult BMI for females at age 40 and 50 y ($r=0$).[37] Nonetheless, obesity among older adults 64–73 y was three times as likely for children who were obese at age 7 y compared to those not obese for both boys and girls.[32] Owing to its earlier onset, childhood BMI may not have as great an impact on adult BMI and body fat as later measures during adolescence.[6,38,39] Our findings also suggest that the effect of early body shape on overweight may be decreased by increasing physical activity, an association that has not been reported before to our knowledge.

Each of the reproductive factors we tested was significantly associated with BMI and obesity among women. Like our finding, menarcheal age was inversely related to BMI among both college-aged women[8] and young adults aged 31 y.[34] Our result is also consistent with a study among women 18–67 y (mean age 41.5 y),[9] which found that total body fat measured using dual energy X-ray absorptiometry was significantly lower among women whose menarche occurred later and the relationship was stronger among premenopausal women than postmenopausal women.[9] In our study, we did not test for interactions with menopausal status since the majority of our sample (84%) was postmenopausal, and we did not detect a significant interaction with reproductive factors and current age. Whereas Freedman and colleagues[40] also observed a significant relation between age at menarche and adult obesity (mean age, 26 y) in the Bogalusa Heart Study, approximately 60–70% of the effect of menarche was due to the influence of child obesity on both early menarche and adult obesity; thus, the authors concluded that the independent effect of menarcheal age on adult obesity has likely been overestimated.

Decisions about when to begin child-bearing impact BMI and obesity. In our study, a woman whose first birth was at age 17–19 y had a 16% increase in odds of overweight and a 30% increase in odds of obesity, while women whose first birth was 23 y or older were at decreased risk. Age at first birth was inversely related to weight gain among both pre- and postmenopausal Chinese women[41] and an early first birth was significantly associated with overweight and body fat among postmenopausal women.[9,10] We also detected a significant interaction between age at first birth and smoking status, where current smokers had the lowest risk of ow/ob at any age of first birth compared to never and previous smokers. It is possible that women smoke as a way to control pregnancy-associated weight gain.

Parity and age at first birth were significantly related to BMI, overweight, and obesity in our study. Although there was a significant linear trend between parity and BMI, only parity of 4 or 5 was significantly related to obesity, but not overweight (compared to parity 2). Parity was independently related to maternal BMI among multiparous women,[42] and postmenopausal women aged 48–58 y were more likely to be overweight with an increased number of births.[11] However, the effect of parity on BMI may be modified by other factors. Women of higher prepregnancy BMI had greater parity-associated weight gain and higher BMI compared to women of lower prepregancy BMI,[19,34,43] although one study found the opposite effect for African-Americans.[19] While we were unable to examine the effects of prepregnancy BMI on ow/ob in our study, we did find an interaction with physical activity. Wolfe *et al*[19] also showed that physical activity, as well as income, education, smoking, marital status, and employment, modified the effect of parity-associated weight gain, suggesting that the relation between parity, BMI, and overweight are complex.

Our study has several limitations. First, BMI may not be the best indicator of adiposity among older adults because of loss of lean body mass and shifts in fat distribution during aging.[44–46] Secondly, in our models we were not able to include diet, which clearly plays an important role in adult BMI and overweight.[47,48] Eating behaviors such as dietary disinhibition and restraint are associated with higher BMI among women,[49] as is depression and anxiety.[50] It is possible that, as with physical activity, the effect of early exposures studied here may be modified by current diet. In addition, we were unable to adjust for prepregnancy BMI and BMI during pregnancy,[32] as discussed above. Parental BMI also plays an important role in the onset and tracking of childhood obesity. The overall risk of obesity is two- to three-fold higher among individuals with a family history of

obesity and children who had two obese parents were at a seven- to eight-fold greater risk of obesity at age 33 y compared to children of parents with normal BMI.[51] Children of two obese parents also showed the strongest tracking of obesity from childhood (7 y) to adulthood (33 y), with correlations of $r = 0.46$ for boys and $r = 0.54$ for girls.[52] The impact of parental obesity on the tracking of childhood obesity into adulthood may exert its effect both through the heritability of BMI[53] and through parental behaviors such as high energy intake and low physical activity.[54] Even so, childhood obesity is an important predictor of adult obesity regardless of whether the parents are obese,[55] and childhood weight was a more important predictor of weight at 20 y than parental weight.[56] Data on diet, prepregnancy BMI, BMI during pregnancy, or parental obesity could alter our study findings.

An additional limitation of our study is whether recalled body shape at age 10 y accurately reflects actual childhood body size and how well this variable correlates with childhood BMI and/or obesity. Although there are data to support the validity of this measure among older women,[15] as noted previously, it remains possible that women who are overweight are more likely to recall their childhood body size as large, and this recall bias would inflate our estimate. A similar bias may also have affected the recall of birthweight. However, our results are consistent with many other reports that have found childhood BMI and birthweight are significant predictors of adult BMI.

In summary, early anthropometric measures and reproductive factors are significantly associated with BMI, overweight, and obesity among older women. The effects of childhood body weight, age at first birth, and parity may be modified by adult lifestyle choices, as well as age. More research is needed to further understand how risk factors that occur early in life may be modified by adult lifestyle choices, since this could have important implications for reducing the burden of adult overweight and obesity.

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